RECOVERY FROM RABIES, WITH REPORTS OF CASES OF TREATMENT PARALYSIS AND OF RECOVERY OF ANIMALS APPARENTLY RABID

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Notwithstanding the occasional reports of natural recoveries from rabies, this disease is generally considered to be invariably fatal. This has no doubt led to some errors in diagnosis, and persons have undoubtedly been exposed to rabies infection without being given the protection of preventive inoculation, while others developing the disease have been given doses of sedatives to control the symptoms, which might in themselves prove fatal. In fact in our own experience we have known of patients receiving doses of sedatives which might easily have been the cause of death.

Unfortunately, owing to obvious difficulties, few of the reported cases of recovery either from natural or experimental infection with rabies virus have been confirmed by subsequent laboratory investigation. Opportunities for such a study are rare. We can find in the literature no cases of recovery after natural infection in man or animals in which the diagnosis was confirmed. In our own experience we have had the three histories (included in this article) given by veterinarians but in each case too long after recovery to permit of confirmatory tests. We now report in detail the study of two cases in animals, and observations on treatment paralysis.

TREATMENT PARALYSIS

This comparatively rare incident of antirabic treatment is made manifest by symptoms that vary in severity from those of a slight sensory neuritis to those of an acute ascending paralysis, which may end fatally. These symptoms usually appear from a week after commencement of treatment to shortly after its completion and occur most often in adults.

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Levy[1] reports one case in which they first manifested themselves 73 days after treatment. Fielder[2] gives the total number of reported cases up to 1916 as 142, with 24 deaths (16.2%). Since then additional reports have been published by Mejio,[3] 24 cases, 4 of which were fatal; Geiger,[4] 8 cases of paralysis and 7 of neuritis; Levy,[5] 1 case; Rochaix and Durand,[5] 1 case; Wirschubski,[6] 1 fatal case; von Dziembowski,[7] 1 case; Papamarku,[8] 9 cases; Pfeiffer,[9] 1 case; Price,[10] 1 case.

To show the relative frequency of its occurrence under different methods of antirabic treatment, Fielder gives the following summary from Simon:

Method	No. of Cases Treated	Cases of Paralysis	Proportion
Classical Pasteur	32,676	6	1:5446
Modified Pasteur	8,657	16	1:541
Högyes	51,417	3	1:17139

Our own experience is summarized in Table 1.

Patients with both the mild and severe forms of paralysis recover with astonishing rapidity in some cases, while in others the course of the disease is protracted and sometimes permanent paralysis results, as in the cases reported by Levy and Price.

Koch[11] believed that treatment paralysis was caused by street virus, modified by the treatment, and supported this claim by reporting the result of inoculation of a number of animals from the lumbar cord of a fatal case. Some of these animals died, after a long incubation period, with symptoms of rabies. This would indicate street virus infection.

When the cord method of preventive treatment is used, paralysis and neuritis are much more common after the intensive than after the milder forms of treatment. Many of the reported cases have occurred in cases with slight injuries. This would seem to us to indicate that the great majority of these cases are due to a fixed virus infection. Kozewalow[12] contended that fixed virus infection caused these symp-

[1] Jour. Am. Med. Assn., 1917, 69, p. 1873.
[2] Jour. Am. Med. Assn., 1916, 66, p. 1769.
[3] Semana Medica, Buenos Aires, 1917, 24, p. 1.
[4] Jour. Am. Med. Assn., 1917, 68, p. 513.
[5] Arch. de Med. Exper., 1916-17, 27, p. 387.
[6] Neurol. Centralbl., 1918, 37, p. 586.
[7] Deutsch. med. Wchnschr., 1916, 42, p. 874.
[8] Ztschr. f. Hyg. u. Infektionskr., 1918, 81, p. 85.
[9] Beitr. z. klin. Wchnschr., 1917-18, 6, p. 87.
[10] Jour. Nerv. & Ment. Dis., N. Y., 1917, 45, p. 67.
[11] Ztschr. f. Hyg. u. Infektionskr., 1909, 64, p. 258.
[12] Centralbl. f. Bakteriol., O., I, 1914, 73, p. 54.

toms, and seemingly proved his contention by citing one case of his own and seven other fatal cases in which portions of the cord and medulla, removed at necropsy and inoculated into rabbits, killed them in from 5 to 8 days, and showed that in incubation period and other characteristics the virus from these cases resembled fixed virus.

TABLE 1

CASES OF TREATMENT PARALYSIS FOLLOWING ANTIRABIC INOCULATIONS

Case	Sex	Age	Character of Injury	Animal	Diagnosis	Time of Onset	Character and Location	Duration and Results
Cord Method. Schedule of Hygienic Laboratory.....1,680 cases								
1	M	30	Hands soiled by saliva	Dog	Laboratory	7 days after completion of treatment	Facial nerve, similar to Bell's palsy	Recovered in 8 days
2*	M	41	Lacerated arm	Dog	Laboratory	7 days after completion of treatment	Right hand and arm, similar to telegrapher's cramp	Recovered in 6 weeks
3	M	36	Hands soiled by saliva	Man	Clinical	8th day of treatment	Painful and tingling sensation on back of thumb and index and middle fingers; some loss of extension	Recovered in 3 mos.
4	F	40	5 punctures on leg and arm	Cat	Laboratory	9 days after completion of treatment	Patient apprehensive and depressed; tingling and very painful sensation on ear, side of face and neck	Almost well in 6 weeks
5	M	40	Lacerated arm	Dog	Laboratory	3 days after completion of treat-	Painful and tingling sensation with almost complete loss of function of entire injured arm; onset accompanied with nervous excitement and apprehension	Partial recovery when last seen 5 weeks after onset
6	F	29	Punctures, arm and forearm	Dog	Laboratory	10th day of treatment	Complete ascending paralysis extending above waist and involving sphincters	Recovery rapid, practically well in 4 weeks
Dilution method. (Approximately 1,000 minimal lethal doses for 13 to 18 days following 3 days dead virus).....2,268 cases treated								
7	M	34	2 punctures on wrist	Dog	Laboratory	8 days after completion of treatment	Severe pain in left side of abdomen and left leg which was partially paralyzed	In bed 1 week. Recovery practically complete in 3 months
Cumming Method. No cases out of.....59 cases treated								

As the symptomatology of these fatal cases, which have been investigated by inoculation of animals, did not differ from that of many severe cases of treatment paralysis that recovered, our conclusion is that at least some of the cases which recover are either infected with street virus of less virulence than usual, or with fixed virus, and in either event represent recoveries from rabies in man.

It should be noted that Hasseltine [13] and others contend that the paralysis is probably due to anaphylaxis from repeated injections of nerve tissue and cite the negative inoculation results of Babes' [14] case to uphold this contention, while others consider it a result of the injection of a hypothetical rabies toxin. These theories may explain certain of the cases, but not all of them, as it is difficult for us to believe that the brain of a person dying of anaphylaxis or from the effects of a toxin, should harbor living and virulent virus.

That treatment paralysis occurs also in animals is shown by a case reported to us by Dr. C. H. Case of Akron, Ohio.

A draft horse, bitten quite severely on the nose, was put under treatment on the fourth day after the injury. The treatment consisted of 3 days' injection of 75 mg. carbolized fixed virus, followed by 7 daily injections of approximately 10,000 minimal infective doses of living fixed virus.

On the 22nd day after the bite and the 18th day after the commencement of treatment the animal became nervous and excitable and refused to take the bit when attempts were made to harness him. He rapidly became weak and incoördinate in his gait, until scarcely able to move without falling. After 3 days' illness improvement began and was so rapid that in about a week the animal was put at light work.

This is the only case of trouble of any sort following antirabic treatment with massive doses of fixed virus, among 247 cases of horses, cows, and hogs, for which we have furnished treatment.

REPORTED RECOVERY IN MAN FROM NATURAL INFECTIONS

We can find no record of recovery in persons who have been infected with rabies by the bites of rabid animals and in whom the diagnosis has been confirmed by laboratory methods, but we wish to call attention to some of the reported recoveries in which the symptoms were so similar to those of hydrophobia that the attending physicians made this diagnosis.

In all the cases reported by Högyes [15] as possible recoveries in man, Pasteur treatment had been given so that treatment paralysis or fixed virus infection cannot be excluded.

In 1913, Moon [16] published a report on 3 inoculated dogs which recovered when bisulphate of quinin had been administered by mouth after the first symptoms had appeared.

[13] U. S. Pub. Health Service, 1913, p. 2220.
[14] Babes and Mironescu: Compt. Rend. Soc. de Biol., 1908, 64, p. 964.
[15] Nothnagels Spez. Path. u. Therap., 1897.
[16] Jour. Infect. Dis., 1913, 13, p. 165.

This stimulated the search for a specific drug and Harris[17] reported an apparent recovery, after the use of quinin, in a man who, after the Pasteur treatment was administered, developed symptoms of rabies, which would have been considered characteristic if he had died. Later experiments by Moon[18] failed to confirm the specific action of quinin reported in his earlier paper. Cumming,[19] Frothingham and Halliday[20] and Coward[21] using rabbits, and Krumweide and Mann[22] using rabbits and dogs, found that quinin did not prevent the appearance of rabies, and Fielder,[23] Geiger,[24] Wesson[25] and others failed to alter in the least the course of developed human rabies by quinin.

Considering the resistance of rabies virus to the action of phenol, one would not expect this chemical to have therapeutic value, but Haberlin[26] reported an apparent cure of rabies in man by the use of phenol injected hypodermically. Fielder tried this method also, but his patient died.

An apparently "hopeless case" of rabies in a 13 year old child recovered when treated by R. Tonin[27] with 0.3 gm. of salvarsan together with KI, tepid baths, and stimulants. But the salvarsan treatment was ineffectual in human cases, when administered by Wesson and L'Arzt.[28] Mejio[29] found it of no benefit in treating man or animals with rabies and also showed that after death their brains were infectious.

With this evidence we must conclude that the specific cure for rabies has not been found. On the other hand, considering the great experience of some of the authors reporting apparent cures, it would seem possible that some of these cases might represent spontaneous cures, such as we herein report, as certainly occurring in one dog, and probably in a cow and in three other dogs.

It is to be hoped that all cases, not evidently hysterical, of persons recovering from an illness resembling rabies, and following the bite of an animal, be reported fully by clinicians, and that whenever possible inoculation into animals of the saliva of these persons should be made; if the person has not received Pasteur treatment his serum should be tested for rabicidal properties, after recovery. It should be remembered that the degree of virulence of the saliva of a rabid man has not been definitely determined, and, as is the case in other animals, is probably extremely variable. Much work remains to be done in regard to rabicidal serum.

[17] Jour. Am. Med. Assn., 1913, 61, p. 1511.
[18] Jour. Infect. Dis., 1915, 16, p. 58.
[19] Ibid., 1914, 15, p. 205.
[20] Jour. Med. Res., 1914, 30, p. 275.
[21] Southern Med. Jour., 1915, 8.
[22] Jour. Infect. Dis., 1915, 16, p. 24.
[23] Jour. Am. Med. Assn., 1916, 66, p. 1300.
[24] Calif. State Med. Jour., 1916, 14, p. 211.
[25] Jour. Am. Med. Assn., 1914, 62, p. 204.
[26] N. Y. State Jour. of Med., 1913, 13, p. 493.
[27] Policlinico, 1912.
[28] L'Art, Wien. klin. Wchnschr., 1917, 30, p. 1515.
[29] Semana Med., 1913, 20, p. 1301.

RECOVERIES IN INOCULATED ANIMALS

Pasteur[30] was the first to establish the fact that recovery occasionally occurred in inoculated dogs after the first symptoms were manifest. Since that time Högyes reported that of 150 successfully inoculated dogs, 13 (8.1%) recovered after being ill with symptoms which he considered characteristic. Six of these had not been given the antirabic treatment.

Joseph Koch[11] reports three spontaneous recoveries of 40 experimentally infected dogs. These 3 had been inoculated with the same strain of street virus.

The case of recovery from rabies in a dog after a successful inoculation with fixed virus recorded by Damon and Hazencamp[31] is of especial interest since the diagnosis was confirmed by the reproduction of the disease in a rabbit inoculated with the saliva of the sick dog. A similar observation by Remlinger[32] is of even greater importance, because he not only confirmed the diagnosis by inoculation of guinea-pigs, while the dog was sick, but continued them after the dog's recovery and obtained positive results as late as 5 days after the dog was completely well. The diagnosis of rabies in the later pigs was confirmed by passage through rabbits.

Almost every worker in antirabic laboratories where fixed virus is standardized by inoculation into rabbits has, like Remlinger,[33] had the experience of seeing a rabbit inoculated with the higher dilutions, show symptoms of rabies and later recover. Koch states that recovery in rats and guinea-pigs has also been noted.

Heretofore it has been customary among physicians and veterinarians in this country to make a negative diagnosis of rabies whenever a man or animal showing rabiform symptoms recovered, but we do not think this course justified in the light of numerous articles which we have cited. Some of the published reports of poliomyelitis and similar conditions following the bite of a dog[34] will bear critical analysis.

RECOVERIES IN ANIMALS AFTER NATURAL INFECTION

We have found reports similar to the first 3 herein recorded, in which there was a clinical history of natural rabies infection in animals with spontaneous recovery, but in which there was no confirmatory laboratory diagnosis. In our case 5 we consider the diagnosis established. In case 4 we consider the serologic findings suggestive.

CASE 1.—The following case was reported by Dr. Walter Brown of Columbus, Ohio, a veterinarian with a large clinical experience in canine diseases, who describes the case thus:

[30] Pasteur's Communication to the Academy, 1882, Expositions 9-10.
[31] Deutsch. tierarztl. Wchnschr., 1908, 16, p. 457.
[32] Compt. Rend. Soc. Biol., 1907, 62, p. 800.
[33] Ann. de l'Inst. Pasteur, 1919, 33, p. 735.
[34] Jour. Am. Med. Assn., 1912, 59, p. 2312.

"A large pointer dog suddenly became sullen and cross, and when seen was so aggressive that a close examination was impossible. He was secured, taken to the hospital, and placed in the rabies ward, where he refused to eat food, but ate his own feces, and seized any foreign bodies presented to him. He was very noisy and had the howl of a rabid dog.

"There was no history of exposure, but rabies was epizootic at that time. After about 3 days his hind quarters partially lost their coördination and his lower jaw showed the usual 'drop jaw' symptom so characteristic of dumb rabies. At the same time he became very weak, his gait was tottering, and his respirations noisy. He was no longer aggressive, but extremely restless, and looked like the ordinary case of paralytic rabies.

"His condition changed but little, excepting that he grew gradually weaker from day to day until about the eighth day, when he began to grow better; on the ninth day he drank a little and took some nourishment. From this time his improvement was steady, so that he was able to walk home, a considerable distance, in 2 weeks from his admission to the hospital. His jaws were still partially paralyzed.

"When seen some months later he had recovered full strength and use of his legs and body, excepting the jaws, which remained weak. A very considerable atrophy of the masseter muscles had occurred, from which the owner reports he never fully recovered."

CASE 2.—This report was made to us by Dr. J. V. Shoemaker, Department of Veterinary Medicine, Ohio State University. "The dog in question, a small fox terrier, was presented for examination Oct. 31, 1918. Eight weeks before, within 2 miles of the dog's home, a rabid dog had bitten 3 horses and a cat. One of the horses bitten was given Pasteur treatment and is now in good condition. The other 2 were not treated and both developed rabies about 5 weeks later. The owner of the patient reported that another terrier owned by him had disappeared 10 days before and was not heard of again.

"At the time of examination the patient had been sick one day and showed a vacant, staring expression, unequal pupils, paralysis of lower jaw and, in fact, all the symptoms which are typical of dumb rabies.

"No treatment was given, but the animal was kept under observation. His condition remained apparently unchanged until the 8th day, when improvement commenced. This continued gradually until Nov. 13, when he was discharged from the hospital and is alive and well at this date, 2 years later."

CASE 3.—This case was reported to us by Dr. Norton Dock, Cincinnati, Ohio. "A setter dog, 3 years of age, with no definite history of exposure, though rabies was prevalent in Cincinnati at the time, developed symptoms of dumb rabies,' such as general depression, paralysis of the lower jaw, dyspnea with its characteristic throat sounds. When he attempted to drink he was unable to swallow. His owner stated that the dog had been sick for several days. He was admitted to the hospital with a diagnosis of rabies. Two days later the dog seemed more animated and the angle between the mandibles was lessened. At this stage it was evident that his efforts to swallow were somewhat successful, both water and liquid food being taken after an effort. The dog rapidly became brighter and stronger so that he was taking solid food in a week's time, and its expression and appearance were practically normal. He was quarantined 14 days longer and dismissed. He was killed by a street car a year later."

CASE 4.—This case was reported to us by Dr. C. H. Case of Akron, Ohio. He was first called to see the cow July 4, 1920, and found her very gaunt and

TABLE 2

EFFECT OF SERUM ON APPEARANCE OF FIRST SYMPTOMS AND COURSE OF DISEASE

Series 1, inoculated 9/9/20, 8 p. m., using fixed virus 328 B mixed with appropriate amount of serum to give dilutions indicated, and held for 30 min. at 37 C. Rabbits used for all tests.

Series 1	Serum Dilution	9/15 A. M.	9/16 A. M.	9/17 A. M.	9/18 A. M.	9/19 A. M.	9/19 P. M.	
	Control (2)	1st Symp.	Sick	Died				
	Undiluted	.....	.....	1st Symp.	Sick	Sick	Died	
	1:2	.....	1st Symp.	Sick	Died			
	1:5	.....	1st Symp.	Sick	Died			
	1:10	.....	.....	.....	.....	.....	.....	Lived
	1:20	.....	.....	1st Symp.	Sick	Sick	Died	
	5 c c intraperitoneally (1)	.....	.....	1st Symp.	Sick	Sick	Died	
	1 c c intraperitoneally (1)	.....	.....	.....	.....	.....	.....	Lived

Series 2, inoculated 9/18/20, 8:30 p. m. using fixed virus 325 A mixed with appropriate amount of serum to give dilutions indicated, and held for 24 hours at 20 C. Rabbits used for all tests.

Series 2	Serum Dilution		9/24 A. M.	9/24 P. M.	9/25 A. M.	9/26 A. M.	9/26 P. M.	9/27 A. M.	9/27 P. M.	9/28 A. M.
	Control (2)		1st Symp.	Sick	Sick	Sick	Died			
	Control guinea-pig serum (3)	A	1st Symp.	Sick	Sick	Sick	Sick	Sick	Died	
		B	1st Symp.	Sick	Sick	Sick	Sick	Died		
	Undiluted	A	1st Symp.	Sick	Sick	Sick	Died			
		B	1st Symp.	Sick	Sick	Died	Sick			
	1:5	A	.....	.....	.....	Very slight symptoms			Lived	
		B	.....	.....	.....	1st Symp.	Sick	Sick	Died	
	1:10	A	.....	.....	.....	.....	.....	.....	.....	Lived
		B	.....	.....	.....	.....	.....	.....	.....	Lived
	1:20	A	.....	.....	1st Symp.	Sick	Sick	Sick	Died	
		B	.....	.....	1st Symp.	Sick	Sick	Died		
	1:50	A	.....	1st Symp.	Sick	Sick	Died			
		B	.....	1st Symp.	Sick	Sick	Died			
	1:100	A	1st Symp.	Sick	Sick	Sick	Died			
		B	1st Symp.	Sick	Sick	Sick	Sick	Died		

Series 3, inoculated 10/1/20, 8 p. m. using virus 323 A mixed with appropriate amount of serum to give dilution indicated, and held for 24 hours at 20 C. Guinea-pigs used for all tests.

Series 3	Serum Dilution		10/5 A. M.	10/6 A. M.	10/7 A. M.	10/8 A. M.	10/9 A. M.	
	Control		.....	.....	1st Symp.	Died		
	1:10	A	Died (traumatic)					
		B	.....	.....	.....	.....	.....	Lived
	1 c c intraperitoneally (1)	A	.....	.....	.....	1st Symp.	Died	
		B	.....	.....	1st Symp.	Sick	Died	

(1) The 2 rabbits in series 1 and 2, guinea-pigs in series 3 received untreated virus intracranially and undiluted serum intraperitoneally. In 2 other guinea-pigs injected similarly Sept. 20, 2.5 c c of serum were ineffective.

(2) For the controls in each series the virus was kept under the same conditions as the virus-serum mixture.

(3) In series 2, 0.5 c c of fresh normal guinea-pig serum was added as a control for any nonspecific serum effect on rabies virus.

not eating and frequently starting and running as though to escape from some terrifying object. The pupils were dilated, and she did not eat or drink but was salivated and drooled over her feed. She had given no milk that morning. Dr. Case instructed the owner to tie her securely in the barn and, avoiding contamination with the saliva, to await developments. The next day she showed the same symptoms but in addition had a peculiar bellow and attempted to attack persons passing before the stall. A diagnosis of rabies was made, and the owner was advised to keep her until she died so that he could claim reimbursement from the county for her loss.

Later it was learned from the owner that the condition remained unchanged for 3 days. Improvement then began and the cow drank and ate a little. In about 10 days the milk flow returned, and she was turned out to pasture. Rabies was prevalent in Akron all this year.

Astonished at such a recovery, Dr. Case reported it to us, and we determined to investigate it.

Dr. J. F. Planz took us to see the cow Sept. 7, and drew for us about 100 c c of blood from the jugular vein. At this time she appeared to be a normal, very placid cow.

The serum from the blood was tested for rabicidal properties (table 2). It will be noted in the 3 series of animals that dilutions of 1 part of serum to 9 parts of normal salt solution were found to have destroyed the infectivity of equal amounts of a 1% solution of fixed virus in salt solution. Slightly larger amounts of serum 0.2 c c to 0.8 c c salt solution (1 part in 5) prolonged the incubation approximately 48 hours, and 1 of 3 rabbits survived. Dilutions of 0.25 c c to 0.48 c (1 in 20) prolonged the incubation about the same length of time. Greater and less amounts of serum were without marked effect.

It is well known that rabicidal serum is often sharply limited in its action, and that the more intensive the immunization, the narrower the zones seem to be. We wish to call attention to the extremely limited zone of activity of this cow's serum, after apparent recovery from rabies.

CASE 5.—The animal, a cocker spaniel, was brought to the University Clinic June 3, 1918, by the owner, who stated that the dog had been bitten about 3 weeks previously by a neighbor's dog which had died soon after. The owner stated that the dog had acted as though choking and that he had kept his mouth partly open for the past 2 days.

The dog was much depressed, the lower jaw drooped, and the expression and voice were characteristic of rabies. He would not attempt to eat or drink. A clinical diagnosis of "dumb rabies" was made, and this was later confirmed by the entire clinical staff. The clinical diagnosis of rabies was further strengthened by the fact that 3 other dogs bitten on the head by the dog that had bitten this spaniel died of rabies after an incubation period of 13 to 15 days. During the 4 days following admission the condition became worse and rabies symptoms more marked. There was partial paralysis of the hind legs so that by the 4th day he was unable to stand. On the 5th day the condition was unchanged, but on the 6th a slight improvement was noticed, and he made efforts to eat. During the next 4 days improvement was plain and on the 10th day he could stand. Improvement continued fairly rapidly except the drooping jaw. After 15 days he could hold up his jaw for a time, but it continued to droop at intervals for 4 weeks. The general condition improved

greatly, and he was apparently normal on the 33d day after admission. He was killed on the 38th day. Inoculations of the brain into 2 rabbits intracranially and into guinea-pigs intramuscularly were negative, and no Negri bodies were found.

On the 9th day of the disease swabs, similar to those used for diagnostic purposes in diphtheria, were inserted in the dog's mouth and were allowed to rest near the opening of the submaxillary duct until saturated. These were then inserted into lacerated cuts in the nape of the neck of 2 young rabbits.

Rabbit A died 6 days later with general paralysis, following convulsions. Rabbit B died with typical symptoms of rabies 20 days after inoculation. The first of these rabbits proved particularly interesting as rabies virus was present in its brain even though the incubation was short and cultures from the brain showed contamination with gram-positive diplococci. By storing in glycerol at —2 C. and inoculating into other animals, a sterile virus was obtained. This was carried through 5 passages as shown in table 3.

The only light which we can throw on the frequency of such recoveries is obtained by going over the records of the Veterinary Clinic, Ohio State University, for 9 years: there are 120 cases in dogs diagnosed as rabies with 2 recoveries, both of which are reported now.

No detailed clinical histories have been included in table 3, but all animals that died as a result of inoculation showed symptoms characteristic of inoculation rabies.

Cultures, not only from the brain but also from the viscera and heart blood, were made in dextrose broth fermentation tubes and on hormone-agar slants in each necropsy in which culture results are noted in the table.

Examinations for Negri bodies were made in smears prepared from Ammon's horn, the cerebral cortex, and the cerebellum. All smears were stained by Williams' method. Only in guinea-pigs 3 and 4 (fourth passage) were large intracellular Negri bodies found typical enough to make a definite positive diagnosis on these alone. In other instances either no bodies were found or, if present, they were so small and of such character that in our routine examination of rabies specimens we would have reported the case "very suspicious." It is worth noting in connection with our experience in demonstrating Negri bodies that Cruikshank and Wright [35] at the Pasteur Institute of Southern India, had quite similar results in their examination of the brains of many animals experimentally inoculated with the saliva of rabid animals. They conclude that "Negri bodies are not always demonstrable in the brains of experimental rabid animals, although they may become so after sub-passage."

[35] Indian Jour. Med. Res., 1914, 1, p. 562.

TABLE 3

RESULTS OF INOCULATION TREATMENT FOR RABIES

Passage 1 Using Saliva of Clinically Rabid Dog, Which Recovered	Passage 2 Using Brain from Rabbit A	Passage 3 Using Brain of Rabbit A_1	Passage 4 Using Brain from Rabbit A_3	Passage 5 Using Brain from Dog 2
Rabbit A. June 12, 1918, inoculated in cut in nape of neck; died June 18, 1918; abscess at site of inoculation, cultures show gram-positive diplococci; smears from brain negative for Negri bodies	Rabbit A_1. July 10, 1918, intracranial; died July 26, 1918; cultures from brain show gram-positive diplococci; smears from brain negative for Negri bodies Rabbit A_2. Dec. 2, 1918, intracranial; died Jan. 27, 1919; cultures negative; smears negative for Negri bodies Dog 6. March 14, 1919, intramuscular; no symptoms by June 30, 1919; dog destroyed Rabbit 6. March 14, 1919, intracranial; cultures negative; smears show small extracellular Negri bodies Rabbit 7. March 14, 1919, intramuscular; no symptoms; discarded after 3 months	Rabbit A_3. Dec. 2, 1918, intracranial; died Dec. 17, 1918; cultures negative; smears negative for Negri bodies Using Brain of Rabbit 6 Rabbit 8. June 12, 1919, intracranial; developed snuffles and abscess of hind leg and was destroyed June 24, 1919 Guinea-pig 1. June 12, 1919, intracranial; died July 13, 1919; cultures negative; small extracellular Negri bodies found Guinea-pig 2. June 12, 1919, intramuscular; died July 8, 1919; cultures negative; small extracellular Negri bodies found	Dog 2. Feb. 10, 1919, intracranial; died Feb. 20, 1919; cultures negative; small extracellular bodies found Dog 4. Feb. 28, 1919, intracranial; no symptoms; destroyed after 3 months Dog 5. Feb. 28, 1919, intramuscular; no symptoms; destroyed after 3 months Using Brain from Guinea-pig 1 Guinea-pig 3. July 13, 1919, intracranial; died Aug. 4, 1919; cultures negative; smears show typical intracellular Negri bodies Guinea-pig 4. July 13, 1919, intracranial; died Aug. 6, 1919; cultures negative; smears show typical intracellular Negri bodies	Dog 3. Feb. 20 1919, intracranial; died Feb. 27, 1919; cultures negative; smears negative for Negri bodies (This animal developed rapid paralysis beginning February 26 and showed a tendency to snap at objects presented to him)

CONCLUSIONS

Spontaneous recovery from rabies naturally acquired, while rare, does occur.

The saliva of an animal which recovers from rabies may have been extremely virulent during the course of the disease.

As early as 38 days after recovery from street rabies in a dog, the infectivity of the brain may disappear and Negri bodies be absent.

Therapeutic measures to control the symptoms in developed rabies in man should not be so heroic as to themselves endanger the life of the patient, for there is a possibility of recovery.